Msx1 and Msx2 function together in the regulation of primordial germ cell migration in the mouse.

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Public Summary:

Scientific Abstract:
Primordial germ cells (PGCs) are a highly migratory cell population that gives rise to eggs and sperm. Much is known about PGC specification, but less about the processes that control PGC migration. In this study, we document a deficiency in PGC development in embryos carrying global homozygous null mutations in Msx1 and Msx2, both immediate downstream effectors of Bmp signaling pathway. We show that Msx1(-/-);Msx2(-/-) mutant embryos have defects in PGC migration as well as a reduced number of PGCs. These phenotypes are also evident in a Mesp1-Cre-mediated mesoderm-specific mutant line of Msx1 and Msx2. Since PGCs are not marked in Mesp1-lineage tracing, our results suggest that Msx1 and Msx2 function cell non-autonomously in directing PGC migration. Consistent with this hypothesis, we noted an upregulation of fibronectin, well known as a mediator of cell migration, in tissues through which PGCs migrate. We also noted a reduction in the expression of Wnt5a and an increase in the expression in Bmp4 in such tissues in Msx1(-/-);Msx2(-/-) mutants, both known effectors of PGC development.